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Functional changes in brain oscillations in dementia: a review

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Abstract: A growing body of evidence indicates that several characteristics of electroencephalography (EEG) and magnetoencephalography (MEG) play a functional role in cognition and could be linked to the progression of cognitive decline in some neurological diseases such as dementia. The present paper reviews previous studies investigating changes in brain oscillations associated to the most common types of dementia, namely Alzheimer's disease (AD), frontotemporal degeneration (FTD), and vascular dementia (VaD), with the aim of identifying pathology-specific patterns of alterations and supporting differential diagnosis in clinical practice. The included studies analysed changes in frequency power, functional connectivity, and event-related potentials, as well as the relationship between electrophysiological changes and cognitive deficits. Current evidence suggests that an increase in slow wave activity (i.e., theta and delta) as well as a general reduction in the power of faster frequency bands (i.e., alpha and beta) characterizes AD, VaD, and FTD. Additionally, compared to healthy controls, AD exhibits alteration in latencies and amplitudes of the most common event related potentials. In the reviewed studies, these changes generally correlate with performances in many cognitive tests. In conclusion, particularly in AD, neurophysiological changes can be reliable early markers of dementia.

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Keywords: Alzheimer disease; brain oscillations; ERP; frontotemporal dementia; vascular dementia.

Introduction

Different types of dementia

Dementia is a clinical syndrome including several and heterogenous disorders of the brain of which, one of the most common is Alzheimer's disease (AD). AD is a progressive neurological disorder characterized by cognitive changes such as memory loss, inability to learn and plan normal activities, and altered perception of space (Castellani et al. 2010). In the brain, AD is characterized by extracellular deposition of amyloid beta peptide, intracellular neurofibrillary tangles of hyperphosphorylated tau protein, neuritic plaques, and progressive and massive loss of neurons and synapses in the cortex (D'Amelio and Rossini 2012). Distinct pathology is associated with other dementing disorders such as frontotemporal degeneration (FTD) and vascular dementia (VaD).

FTD is a heterogeneous disorder with several clinical phenotypes associated with multiple neuropathologic entities. In its most common behavioural manifestation, FTD includes personality changes, altered executive functions and, in later stages, non-fluent aphasia (Neary et al. 2005). Behavioral changes include decline in social interpersonal conduct, impaired regulation of personal conduct, emotional blunting, loss of insight, mental rigidity, and inflexibility (Bathgate et al. 2001). The most common pathology of FTD is neuronal loss and gliosis affecting the superficial cortical lamina (Mohandas and Rajmohan 2009).

Finally, vascular related brain lesions are heterogenous, leading to a variety of cognitive deficits. Therefore, in this case, the clinical manifestation is typically in a stepwise fashion, and the progression depends on the brain regions affected by the pathological process (Robillard 2007). VaD symptoms range from deficits in attention, information processing, and executive functions (O'Brien and Thomas 2015) to other deficits such as aphasia and apraxia (Koga et al. 2009).

The role of brain oscillations in the diagnosis of dementias

Recent studies have shown that cognitive decline in neurodegenerative diseases are accompanied by alterations in brain oscillatory activity (Nimmrich et al. 2015). Studies conducted in animals have shown that oscillatory activity modulates the timing of neuronal spiking, allowing the communication among distributed cortical areas, and it has a functional role in cognitive processes (Fries 2005; Jacobs et al. 2007). Therefore, different patterns of alteration in brain rhythms might underlie different types of cognitive deficits. Previous studies have reported changes in the power of specific frequency bands in dementia. In general, a power increase has been found in the slower frequency bands (Huang et al. 2000) as well as a decrease in the faster frequency bands (Kikuchi et al. 2002). Changes have also been reported in both latency and amplitude of event-related potentials (ERP), especially in patients with AD (Vecchio et al. 2014). Interestingly, some of these studies reported correlation between changes in brain oscillations and cognitive deficits (Gianotti et al. 2007).

As abnormalities in brain rhythms may be present since the early stages of the disease (Laske et al. 2015), they may constitute early markers for diagnosis. A previous review focused on brain oscillations in AD with the aim of identifying a marker for early diagnosis (Jafari 2020). However, no studies have reviewed the literature and compared AD with other types of dementia. Early differential diagnosis might allow both the use of recent therapeutic techniques and the timely information for patients and caregivers. Additionally, making a clear diagnosis allows to direct patients to adequate support networks guaranteeing an early access to treatment, appropriate information and support to improve quality of life. Patients can be involved in decision-making processes to plan their future choices and activities. Depending on the dementia's type, practitioners can predict which symptoms will develop first to adapt the patient's activities based on this knowledge and to delay as much as possible the patient's hospitalization, which is a major cost driver in dementia.

To date, diagnosis of dementia is a lengthy and complex process needing multiple assessment involving longitudinal observation, neuropsychological evaluation as well as the use of neuroimaging techniques and, especially for AD, the use of other biomarkers. In particular, measures of cerebrospinal fluid (CSF) levels of Aβ and tau, and magnetic resonance imaging (MRI) measures of brain grey matter volume are considered as major biomarkers (D'Amelio and Rossini 2012; Hohman et al. 2017). However, these techniques are rather specific and costly and many care centers do not hold them. Additionally, no clear biomarkers have been identified for other types of dementia. Therefore, a high rate of underdiagnosis of dementia has been reported in primary care (Boise et al. 1999). Finding a pattern of changes in brain oscillations characterizing each type of dementia could support early differential diagnosis, guide treatments, and consequently reduce care costs. Such changes may be detected by using magnetoencephalography (MEG) and electroencephalography (EEG). MEG is a relatively expensive device, whereas EEG is a widely used, relatively low-cost, noninvasive tool that could be employed together with other biomarkers for the detection of brain functional abnormalities in the early stage of dementia. As cognitive deficits are prominent in dementia, investigating the correlation between brain rhythms and cognitive performance would allow clarifying to what extent cognitive deficits may be predicted by changes in electrophysiological activity. Although to date there is no definitive cure for dementia, early differential diagnosis and therapeutic interventions aimed at restoring brain oscillations in the early stage of the disease could delay the onset of cognitive deficits and the progression of symptoms (Hsiao et al. 2013).

Spontaneous and stimulus-driven brain oscillations

Each of the components in which electrical signals from neurons can be separated and analysed conveys peculiar physiological information on brain functional state (Vecchio et al. 2014) that can be classified in the time-frequency domain to study oscillatory activity or in the time domain to study ERP (Hauk et al. 2006). Oscillatory activity can be classified as ongoing/spontaneous or event-related. In the analysis of spontaneous EEG, only sporadic changes of amplitudes from hidden sources are measured (Başar et al. 2013). Conversely, event-related activity can be evoked or induced, and it is elicited by endogenous as well as by exogenous stimuli. Induced responses probably reflect top-down modulation and they are non-phase locked responses with variations in latency with respect to the stimuli (Chen et al. 2012). Evoked responses are generally locked to the stimulus presentation and linked to bottom-up processes. Therefore, increasing cognitive demand such as attention, memory and perception leads mostly to enhancement of induced responses (Chen and Huang 2016).

Brain oscillations are traditionally classified into five frequency bands: delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), beta (12–35 Hz), and gamma (>35 Hz). Different functional roles have been reported for each frequency band (Jensen et al. 2019). In particular, delta oscillations are preeminent during those attention processes in which external stimuli have to be anticipated (Lakatos et al. 2008), in signal detection and decision making (Başar-Eroglu et al. 1992), theta oscillations are associated with memory (Scheeringa et al. 2009) and error processing (Luu et al. 2004). Alpha rhythm has been shown to play a role in attention and visual processes (Bonnefond and Jensen 2012) as well as in working memory maintenance (Palva and Palva 2011). Beta oscillations are strongly associated with the motor system and reflect sensory motor processing (Van Ede et al. 2011) as well as motor decision making (Donner et al. 2009). Finally, gamma rhythm is typically associated with neuronal processing of information, memory maintenance (Gruber et al. 2004; Jokisch and Jensen 2007), and attention (Bauer et al. 2006).

Generally, slow frequencies reflect long-range communication between brain regions, whereas higher frequencies reflect local communication between neural populations (Siegel et al. 2012). Such activity can be measured in terms of absolute or relative power. Absolute power is the integral of all power values within its frequency range (Yuvaraj et al. 2014) that represents the total energy intensity of an electrode in a certain region of the brain (Cunha et al. 2004). Relative power is calculated by dividing the absolute power of each frequency band by the total power of the whole spectrum in all bands (John et al. 1988). In resting state condition, each of these frequency bands is present primarily in specific regions of the brain (Sanei and Chambers 2013). Namely, slower waves, such as delta and theta, can be observed mainly in medial frontal areas (Harmony 2013). The activity of the alpha band is distributed mainly in the occipital and parietal regions and the beta and gamma oscillations are prominent in the frontal and central areas (Sanei and Chambers 2013). However, this region-specific localization should be taken cautiously as brain oscillations depend on complex neural dynamics that cannot be segregated in specific brain areas. Abnormalities in resting state brain oscillations have been found in several neurological and psychiatric conditions (Başar et al. 2013).

Finally, EEG activity in the time domain measures ERP, electrical potentials generated by the brain in response to different internal or external events. There are several ERP components, obtained using different paradigms that provide information about many cognitive processes (Luck 2012). Early ERP peaking within one hundred milliseconds after stimulus onset are considered exogenous components representing sensory processes as they depend mainly on the characteristics of the stimulus (Sur and Sinha 2009). Among ERP components, the auditory N100 is usually enhanced in attention shift (Katada et al. 2005), P200 is linked to attention and discrimination (Conley et al. 1999), N200 can be related to selective attention and storage of sensory information (Sokhadze et al. 2017) and P300 is linked to attention during memory and to responses' orientation (Katada et al. 2005) and stimulus categorization (Gentili et al. 2014). The amplitude of the evoked potential changes depending on the attention load (Sur and Sinha 2009) with a smaller amplitude reflecting a smaller amount of attention to the stimulus. Longer ERP latency is associated with a greater difficulty in discriminating stimuli (Sur and Sinha 2009).

Aims of the present review

The present study intended to review the latest evidence reporting resting-state EEG or MEG changes in AD, FTD, and VaD in terms of frequency power, coherence, synchronization, and ERP. Specific aims are: 1) to identify functionally relevant changes in oscillatory brain activity in the traditional frequency bands and their link to specific brain regions; 2) to investigate principal differences in latency and amplitude of the ERP components; 4) to gather results about the relationship between EEG/MEG changes and cognitive deficits reported in the included studies. Finally, possible mechanisms underlying changes in brain oscillations in dementia are discussed.

Methods

Search strategy and study selection

This systematic review was conducted following the Preferred Reporting of Systematic Reviews and Meta-Analyses (PRISMA) statement.

We searched in the following online databases for published articles: SCOPUS and PUBMED from 01/2000 to 01/2021. Namely, we used the following keywords: "Dementia" and "electroencephalography" or "EEG" and "magnetoencephalography" or "MEG" and "brain oscillations" or "brain rhythms". We also screened bibliography of previous reviews.

To be included, candidate studies had to meet the following criteria:

- Participants recruited were diagnosed with one of the following:
  a) Alzheimer's disease, diagnosed by using the National Institute of Neurological and Communicative Disorders and Stroke and Alzheimer's Disease and Related Disorders

Association (NINCDS-ADRDA) criteria and/or by using the DSM criteria;
b) Fronto-temporal degeneration diagnosed according to Neary et al. (1998);
c) Vascular dementia, diagnosed in accordance with the criteria of the National Institute of Neurological Disorders and Stroke Association Internationale pour la Recherche et l'Enseignement en Neurosciences (NINDS-AIREN).

– Using EEG or MEG to measure changes in frequency power, coherence, synchronization or ERP.

To be included, studies were not requested to report cognitive performance, however, when present, cognitive data were taken into account to review the role of brain oscillations in cognitive performance. Candidate studies were excluded if: a) they were aimed at testing the accuracy of the methods rather than changes in brain oscillations, b) they did not use the target measures as outcomes, c) the experimental group counted less than 15 participants, or d) they tested the effect of a medication (Figure 1).

Data extraction

Four authors independently screened the title and abstracts of the articles collected from the database search. Only articles that met the inclusion criteria were selected.

Selected articles were read by two authors that extracted relevant information following a modified version of PICO guidelines: participants, methodologies (instead of interventions), comparisons, and outcomes. Additionally, data relative to sample characteristics were extracted. Frequency power, coherence and synchronization were assessed for each frequency band (delta, theta, alpha, beta, and gamma). Event-related potentials outcomes were investigated for the following components: N100, N200, P200, and P300. Correlations with neuropsychological tests were extracted for frequency power, coherence, and synchronization as well as for ERP.

Results

Included studies: main characteristics

A total of 58 studies, including 2851 patients, met the inclusion criteria. One study enrolled participants with vascular dementia (van Straaten et al. 2012), 47 studies enrolled participants with Alzheimer's disease (Adler et al. 2003; Ally et al. 2006; Ashford et al. 2011; Babiloni et al. 2006a,b, 2007a,b, 2011a,b, 2013a,b, 2015, 2016; Bennys et al. 2007; Canuet et al. 2012; Caravaglios et al. 2010, 2008;

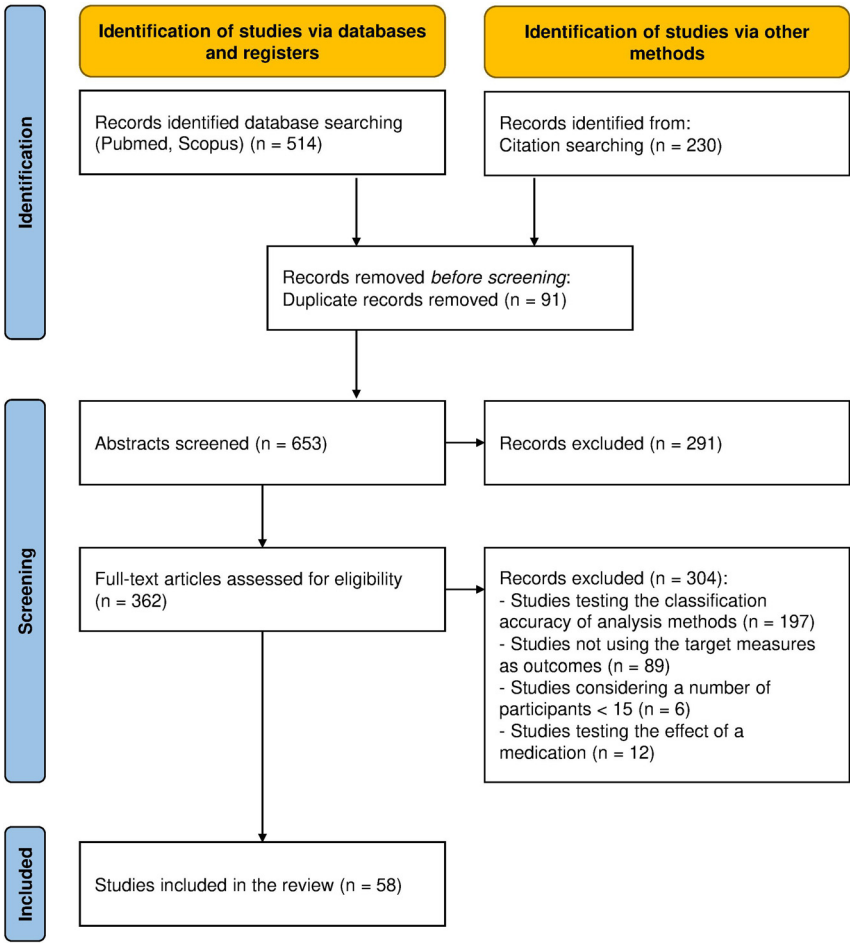

Figure 1: Search strategy used for selection of studies included in the review.

Chang et al. 2014; Chen and Huang 2016; de Haan et al. 2008; Engels et al. 2016; Fernández et al. 2003; Fonseca et al. 2011a,b; Frodl et al. 2002; Gianotti et al. 2007; Gironell et al. 2005; Hata et al. 2016; Hirata et al. 2000; Juckel et al. 2008; Kikuchi et al. 2002; Kim et al. 2012; Knott et al. 2000, 2001; Kurita et al. 2010; Lai et al. 2010; Lee et al. 2013; Lizio et al. 2015; Musaeus et al. 2019; Ponomareva et al. 2003; Poza et al. 2007; Stam et al. 2006, 2002; Sumi et al. 2000; van der Hiele et al. 2007; Vecchio et al. 2014; Zheng-Yan 2005). Four studies compared Alzheimer's disease with fronto-temporal degeneration (Caso et al. 2012; Lindau et al. 2003; Nishida et al. 2011; Yu et al. 2016) and five studies compared Alzheimer disease with vascular dementia (Babiloni et al. 2004b,a; Muscoso et al. 2006; Wu et al. 2014; Yamaguchi et al. 2000). With respect to methodology, six studies used MEG, while 52 studies used EEG. Twenty-eight studies measured frequency power (Babiloni et al. 2007a,b, 2004a, 2006a,b, 2011a,b, 2013a,b, 2015, 2016; Caso et al. 2012; de Haan et al. 2008; Engels et al. 2016; Fernández et al. 2003; Gianotti et al. 2007; Huang et al. 2000; Kikuchi et al. 2002; Kim et al. 2012; Knott et al. 2001; Lindau et al. 2003; Lizio et al. 2015; Nishida et al. 2011; Ponomareva et al. 2003; Poza et al. 2007; Van Straaten et al. 2012; Vecchio et al. 2014; Wu et al. 2014).

Fifteen studies focused on auditory ERP (Ally et al. 2006; Ashford et al. 2011; Bennys et al. 2007; Caravaglios et al. 2010, 2008; Chang et al. 2014; Frodl et al. 2002; Gironell et al. 2005; Hirata et al. 2000; Juckel et al. 2008; Lai et al. 2010; Muscoso et al. 2006; Sumi et al. 2000; Yamaguchi et al. 2000), whereas one study measured visual event-related potentials (Kurita et al. 2010); six studies measured coherence (Adler et al. 2003; Knott et al. 2000; Musaeus et al. 2019; Sankari et al. 2011; Yu et al. 2016; Zheng-Yan 2005), and three studies analysed synchronization (Babiloni et al. 2004b; Hata et al. 2016; Stam et al. 2002). Three studies measured both frequency power and coherence (Fonseca et al. 2011a, 2011b; van der Hiele et al. 2007), one study measured both frequency power and synchronization (Canuet et al. 2012), and another one measured coherence and synchronization (Stam et al. 2006). Regarding cognitive outcomes, 30 studies analysed the correlations between neurophysiological measures and performance in neuropsychological tests (Adler et al. 2003; Babiloni et al. 2007a,b, 2013b,a, 2011b, 2006b, 2004b; Bennys et al. 2007; Caso et al. 2012; de Haan et al. 2008; Fonseca et al. 2011a; Gianotti et al. 2007; Hata et al. 2016; Hirata et al. 2000; Juckel et al. 2008; Kikuchi et al. 2002; Kim et al. 2012; Knott et al. 2000, 2001; Lee et al. 2013; Lizio et al. 2015; Musaeus et al. 2019; Muscoso et al. 2006; Nishida et al. 2011; Stam et al. 2006; van der Hiele et al. 2007; van Straaten et al. 2012; Wu et al. 2014; Yamaguchi et al. 2000). In particular, these studies assessed the correlation between EEG/MEG changes and global cognition (MMSE), verbal memory (Rey auditory verbal learning test, prose memory, word list recall test, digit span test), visuo-spatial memory (Corsi block-tapping test, figure Rey recall test), attention, executive function (Trail making test, Stroop test), or language (word fluency for category, token test).

Discussion

Changes in frequency power

Absolute and relative power alterations in almost all the frequency bands have been reported with normal aging. In particular, aging is associated with increased delta and theta frequencies in parietal and fronto-central areas, respectively, as well as with a reduction in the alpha activity in parietal, occipital, and temporal regions and an increased level of slower delta and theta frequency (Cummins and Finnigan 2007). Most of the studies reviewed here investigated differences in absolute and relative power between AD and healthy participants and between VaD or FTD and healthy participants (Table 1a and b).

All the types of dementia seem to be associated with a general power increase in the lower frequencies and a power decrease in the higher frequency bands.

Increase in delta and theta power in the resting state of AD patients

Previous studies investigating changes in AD reported that, compared to FTD (Lindau et al. 2003), VaD (Wu et al. 2014), and healthy controls (Poza et al. 2007), AD patients exhibit the highest increase in delta and theta power in the resting state (Fonseca et al. 2011b; Huang et al. 2000; Knott et al. 2001). It should be noted that the delta power increases in several regions of the brain (Adler et al. 2003; Babiloni et al. 2013b; Bennys et al. 2007; Canuet et al. 2012; Caravaglios et al. 2010, 2008; Chang et al. 2014), while theta enhancement is mostly localized in the central and occipital areas (Caso et al. 2012; Kim et al. 2012; Nishida et al. 2011).

Possible mechanisms associated

In animal studies, the cholinergic system has been shown to modulate the spontaneous cortical activity in the theta and delta frequency bands (Platt and Riedel 2011). This system has abnormal functioning in AD patients (Giacobini

Table 1a: Characteristics of studies exploring changes in EEG frequency power in Alzheimer's diseases (AD), frontotemporal degeneration (FTD), and vascular dementia (VaD).

Study	Participants characteristics		Main results		
	Population	Comparator	Bands	Changes	Localization
Babiloni et al. (2004a)	AD	Healthy	Delta	Increased	Widespread
			Theta	No changes	
			Alpha	Decreased	Parieto–occipital
			Beta	No changes	
	VaD	Healthy	Delta	Increased	Widespread
			Theta	Increased	Widespread
			Alpha	Decreased	Parieto–occipital
			Beta	No changes	
Babiloni et al. (2006a)	AD	Healthy	Delta	Increased	Occipital
			Theta	No changes	
			Alpha	Decreased	Parieto–occipital, temporal
			Beta	No changes	
Babiloni et al. (2006b)	AD	Healthy	Delta	Increased	Widespread
			Theta	No changes	
			Alpha	Decreased	Temporal, parieto–occipital
			Beta	No changes	
Babiloni et al. (2007a)	AD	Healthy	Delta	Increased	Widespread
			Theta	Increased	Widespread
			Alpha	Decreased	Parietal, temporal, occipital
			Beta	No changes	
Babiloni et al. (2007b)	AD	Healthy	Delta	Increased	Occipital, temporal
			Theta	No changes	
			Alpha	Decreased	Occipital, temporal
			Beta	No changes	
Babiloni et al. (2011a)	AD	Healthy	Delta	No changes	
			Theta	No changes	
			Alpha	Decreased	Parietal, temporal, occipital
			Beta	No changes	
Babiloni et al. (2011b)	AD	Healthy	Delta	Increased	Widespread
			Theta	No changes	
			Alpha	Decreased	Parietal, temporal, occipital
			Beta	No changes	
Babiloni et al. (2013a)	AD	Healthy	Delta	Increased	Widespread
			Theta	No changes	
			Alpha	Decreased	Parieto–occipital
			Beta	No changes	
Babiloni et al. (2013b)	AD	Healthy	Delta	Increased	Central, parietal, temporal
			Theta	No changes	
			Alpha	Decreased	Parietal, temporal, occipital
			Beta	No changes	
Babiloni et al. (2015)	AD	Healthy	Delta	No changes	
			Theta	No changes	
			Alpha	Decreased	Parieto, temporal, occipital
			Beta	No changes	
Babiloni et al. (2016)	AD	Healthy	Delta	Increased	Widespread
			Theta	No changes	
			Alpha	Decreased	Widespread
			Beta	Decreased	Posterior
Canuet et al. (2012)	AD	Healthy	Delta	Increased	Fronto–central
			Theta	No changes	
			Alpha	Decreased	Parieto–occipital
			Beta	No changes	

Table 1a: (continued)

Study	Participants characteristics		Main results		
	Population	Comparator	Bands	Changes	Localization
Caso et al. (2012)	AD	Healthy	Delta	Increased	Posterior
			Theta	Increased	Widespread
			Alpha	Decreased	Widespread
			Beta	Decreased	Temporo–central
	FTD	Healthy	Delta	No changes	
			Theta	Increased	Centro–posterior
			Alpha	No changes	
			Beta	No changes	
Fonseca et al. (2011a)	AD	Healthy	Delta	Increased	Frontal, central, occipital
			Theta	Increased	Widespread
			Alpha	No changes	
			Beta	No changes	
Fonseca et al. (2011b)	AD	Healthy	Delta	Increased	Left hemisphere
			Theta	Increased	Widespread
			Alpha	No changes	
			Beta	No changes	
Gianotti et al. (2007)	AD	Healthy	Delta	Increased	Parietal, temporal, occipital
			Theta	Increased	Widespread
			Alpha	Decreased	Frontal, temporal, occipital
			Beta	Decreased	Widespread
Huang et al. (2000)	AD	Healthy	Delta	Increased	Temporal, fronto–central, centro–parietal
			Theta	Increased	Temporal, fronto–central, centro–parietal
			Alpha	Decreased	Temporal, temporo–occipital, centro–parietal
			Beta	Decreased	Temporo–occipital, centro–parietal
Kikuchi et al. (2002)	AD	Healthy	Delta	No changes	
			Theta	No changes	
			Alpha	Decreased	Occipital
			Beta	Decreased	Occipital
Kim et al. (2012)	AD	Healthy	Delta	No changes	
			Theta	Increased	Frontal, right central, occipital
			Alpha	No changes	
			Beta	Decreased	Frontal, central, occipital
Knott et al. (2001)	AD	Healthy	Delta	Increased	Parietal, temporal, occipital
			Theta	Increased	Widespread
			Alpha	No changes	
			Beta	No changes	
Lindau et al. (2003)	AD	Healthy	Delta	Increased	Global field power
			Theta	Increased	Global field power
			Alpha	Decreased	Global field power
			Beta	Decreased	Global field power
	FTD	Healthy	Delta	No changes	
			Theta	No changes	
			Alpha	Decreased	Global field power
			Beta	Decreased	Global field power
Lizio et al. (2015)	AD	Healthy	Delta	Increased	Widespread
			Theta	Decreased	Frontal
			Alpha	Decreased	Widespread
			Beta	No changes	
Nishida et al. (2011)	AD	Healthy	Delta	Increased	Widespread
			Theta	Increased	Temporal, occipital
			Alpha	Decreased	Parietal, temporal, occipital
			Beta	Decreased	Parietal
				Increased	Frontal
	FTD	Healthy	Delta	No changes	
			Theta	Increased	Posterior temporal
			Alpha	Decreased	Fronto–temporal
				Increased	Parietal
			Beta	Increased	Frontal, parietal, temporal

Table 1a: (continued)

Study	Participants characteristics		Main results		
	Population	Comparator	Bands	Changes	Localization
Ponomareva et al. (2003)	AD	Healthy	Delta	Increased	Not reported
			Theta	Increased	Not reported
			Alpha	Decreased	Not reported
			Beta	Decreased	Not reported
Van Straaten et al. (2012)	VaD	Healthy	Delta	Increased	Not reported
			Theta	Increased	Not reported
			Alpha	Decreased	Parieto–occipital
			Beta	Decreased	Frontal, parieto–occipital
Van Der Hiele et al. (2007)	AD	Healthy	Delta	Increased	Not reported
			Theta	Increased	Not reported
			Alpha	No changes	
			Beta	No changes	
Vecchio et al. (2014)	AD	Healthy	Delta	Increased	Frontal, temporal
			Theta	No changes	
			Alpha	Decreased	Parietal, occipital
			Beta	No changes	
Wu et al. (2014)	AD	Healthy	Delta	Increased	Temporal, parietal, occipital
			Theta	Increased	Widespread
			Alpha	Decreased	Frontal, parietal, occipital
			Beta	Decreased	Widespread
	VaD	Healthy	Delta	No changes	
			Theta	Increased	Widespread
			Alpha	No changes	
			Beta	No changes	

Table 1b: Characteristics of studies exploring changes in MEG frequency power in Alzheimer's diseases (AD), frontotemporal degeneration (FTD), and vascular dementia (VaD).

Study	Participants characteristics		Main results		
	Population	Comparator	Bands	Changes	Localization
Engels et al. (2016)	AD	Healthy	Delta	Increased	Posterior parietal, medio–temporal, occipital
			Theta	Increased	Widespread
			Alpha	Decreased	Parietal, occipital
			Beta	Decreased	Frontal
Fernandez et al. (2003)	AD	Healthy	Delta	Increased	Parietal, left temporal
			Theta	Increased	Parietal, temporal
			Alpha	No changes	
			Beta	No changes	
de Haan et al. (2008)	AD	Healthy	Delta	Increased	Parietal, temporal, occipital
			Theta	No changes	
			Alpha	Decreased	Widespread
			Beta	Decreased	Widespread
Poza et al. (2007)	AD	Healthy	Delta	Increased	Not reported
			Theta	Increased	Not reported
			Alpha	Decreased	Not reported
			Beta	Decreased	Not reported

2003). In particular, the loss of hippocampal and cortical neurons impinged by cholinergic input might be responsible for the enhancement of delta and theta power, especially in the central regions. Degeneration in the mesial temporal cortex might, in turn, affect the functional connectivity between these regions, the hippocampal formation and the posterior brain areas (Killiany et al. 1993) leading to an enhancement of slow wave activity in

posterior brain regions. Moreover, a reduction of entorhinal volumes has recently been correlated with an increment of cortical delta rhythm (Fernández et al. 2003).

Different pathophysiological mechanisms might be responsible for the enhancement in slow wave activity observed in patients with VaD (Babiloni et al. 2004b; van Straaten et al. 2012) and FTD (Caso et al. 2012; Kim et al. 2012; Nishida et al. 2011). For instance, vascular lesions might be involved in the causal mechanisms of loss of connectivity that lead to the slowing of EEG activity (van Straaten et al. 2012). Additionally, since slow wave activity reflects the activity of deeper cortical layers that innervate superficial cortical layers (Buffalo et al. 2011), the cortico–subcortical disconnection of FTD might be responsible for the enhancement of slow oscillations in these patients. However, more studies would be needed investigating the pathophysiological mechanisms involved in abnormal slow wave activity in FTD.

Reduced alpha and beta power in all types of dementia

Alpha and beta power are reduced in AD (de Haan et al. 2008; Engels et al. 2016; Kikuchi et al. 2002; Knott et al. 2001; Lindau et al. 2003; Ponomareva et al. 2003; Poza et al. 2007), as well as in FTD (Lindau et al. 2003) and VaD (van Straaten et al. 2012) compared to healthy controls. In general, the reduction in the alpha band is located mainly in the parietal, occipital, and temporal regions in AD (Babiloni et al. 2013b; Bennys et al. 2007; Canuet et al. 2012; Caravaglios et al. 2010, 2008; Kim et al. 2012; Kurita et al. 2010; Lai et al. 2010; Lee et al. 2013; Lizio et al. 2015) and it has a frontal distribution in FTD (Nishida et al. 2011) and a parieto–occipital distribution in VaD (Babiloni et al. 2004a; van Straaten et al. 2012; Vecchio et al. 2014). This reduction correlates with the level of atrophy of brain grey or white matter (Babiloni et al. 2006a,b, 2013b; Fernández et al. 2003). In particular, a reduction in the alpha power over the occipital lobes correlates with reduction in grey matter density in the occipital areas of the brain in AD (Babiloni et al. 2015). Interestingly, patients carrying the APOE-4 allele exhibit a stronger reduction in alpha oscillations compared to noncarriers (Canuet et al. 2012).

Possible mechanisms associated

A mechanism that might account for the reduction in alpha and beta oscillations is the abnormal functioning of the cholinergic system. Cholinergic basal forebrain pathways drive thalamo–cortical and cortico–cortical loops converging to cortical pyramidal neurons that generate alpha oscillations. AD patients have an impairment of cholinergic basal forebrain and hippocampus (Huang et al. 2000) that might be responsible for the alteration of the alpha rhythm. This would account for the partial restoration of this alteration observed after long-term cholinergic therapy (Babiloni et al. 2006b).

The reduction in beta band is located in frontal, central, and occipital regions in AD (Kim et al. 2012), in frontal and parietal regions in FTD (Nishida et al. 2011) and in frontal regions in VaD (Babiloni et al. 2004b; van Straaten et al. 2012). However, a stronger beta reduction has been observed in AD patients (Nishida et al. 2011). In the motor cortex, activity in the beta band is related to the level of a specific neurotransmitter, that is, the γ-aminobutyric acid (GABA) (Gaetz et al. 2011), so that altered beta oscillations could reflect a dysfunction of the GABAergic system (Hall et al. 2011). Some previous studies have found altered GABA levels in AD [see (Lanctôt et al. 2004) for a review] and in FTD (Adams et al. 2021; Murley et al. 2021). Future studies would be needed investigating whether changes observed in beta oscillations in AD and FTD could be linked to such alterations in GABA levels.

Changes in gamma oscillations in AD and FTD

Gamma oscillations have been found to be reduced in both AD (Herrmann and Demiralp 2005) and FTD (Hughes et al. 2018). Findings relative to gamma oscillations are controversial (Sanei and Chambers 2013). Indeed, some studies reported reduced gamma power and synchronization (Koenig et al. 2005; Ribary et al. 1991; Stam et al. 2002) whereas other studies reported increased gamma oscillations in dementia (Başar et al. 2017). Methodological differences among studies might explain some of the discrepancies (Başar and Düzgün 2016). However, disruption in GABAergic interneuron networks might account for the imbalance between excitation and inhibition and drive a pathological increase in the power of the gamma band (Giovannetti and Fuhrmann 2019). To date, gamma oscillations are less investigated than other frequency bands (Whitham et al. 2007), possibly due to the fact that scalp EEG above 20 Hz is heavily influenced by electromyographic (EMG) activity that is generally filtered out as artefact during EEG analysis. As interest towards the role of the disruption of gamma oscillations in dementia is growing, further studies, able to analyse gamma oscillations while

removing EMG artefacts would be needed. Of special interest would be studies investigating changes in gamma oscillations among different types of dementia.

Overall, changes in brain oscillations might depend on the accumulation of amyloid beta and tau protein aggregates (Muller and Schwartz 1978) as well as on atrophy (Moretti et al. 2012) or neurotransmitters dysfunctions (Murley and Rowe 2018). However, it is not clear how different pathophysiological processes might induce similar patterns of changes in brain oscillations. More studies would be crucial to clarify the pathophysiological mechanisms involved in abnormal brain rhythms.

Correlations between changes in frequency power and cognitive processes

Correlational studies show that abnormalities in cortical brain oscillations could be related to cognitive test performances (Babiloni et al. 2006a). Cognitive processes require long-range and short-range coordination of brain activity over time (Cannon et al. 2014). Although there is no one-to-one mapping between a given oscillatory activity and a single cognitive process, different frequencies might play an important role during specific cognitive activities (Jensen et al. 2019). For instance, a general decrease in alpha power indexes an increasing demand of attention, increasing task load (Van Diepen et al. 2019), as well as an active inhibition of irrelevant sensory processing (Jensen and Mazaheri 2010). Similarly, the beta rhythm is associated with the top–down control of information processing (Jensen et al. 2015). In frontal regions, theta and gamma power are enhanced during working memory tasks (Scheeringa et al. 2009), whereas in medial temporal lobes, theta, and gamma activity has been associated with episodic memory (Herweg et al. 2020). Gamma oscillations are involved in information processing and integration of perceptual information of upcoming stimuli (Jensen et al. 2014).

Most of the studies investigating changes in brain oscillations and cognitive tasks for diagnosis of dementia focused on AD. Overall, in these patients there is a strong negative correlation between slow waves activity and the performance in neuropsychological tests whereas the correlation is positive for fast waves activity (Fonseca et al. 2011a; Kikuchi et al. 2002). In particular, enhanced delta power in the occipital cortex as well as enhanced theta power in frontal, central, and occipital regions negatively correlate with the global cognitive status as measured with the Mini Mental State Examination (MMSE) (Babiloni et al. 2006a, 2016; Gianotti et al. 2007; Kim et al. 2012; Knott et al. 2001; Wu et al. 2014). Conversely, a positive correlation has been reported between alpha and beta power and MMSE scores (Gianotti et al. 2007; Kim et al. 2012). The enhancement in the alpha frequency is mainly located in the parietal, temporal, and occipital cortices (Babiloni et al. 2006a, 2013a,b; Lizio et al. 2015) whereas beta power correlation has been found in the occipital cortex (Kikuchi et al. 2002).

In addition to global cognitive status, correlations have also been found between brain oscillations and cognitive tests that measure attentional, visuospatial, and memory performance (Babiloni et al. 2007b; Fonseca et al. 2011a; Kim et al. 2012). In particular, enhanced power in the delta and theta frequency bands in AD is associated with negative performances in memory, verbal fluency and constructional practice (Fonseca et al. 2011a).

On the other hand, in AD patients, enhanced beta power is positively correlated with attention, memory, visuospatial abilities, and executive functions (Kim et al. 2012).

These findings suggest that whether, on the one hand, a diffuse increase in theta oscillations reflects a general cognitive decline, enhanced beta power in specific brain regions, such as those involved in memory processes, supports preserved performances.

Unfortunately, we found only one study investigating the correlations between brain oscillations and cognitive functions in FTD (Caso et al. 2012) and two studies focused on VaD (van Straaten et al. 2012; Wu et al. 2014). In patients with FTD, alpha cortical sources positively correlate with performance on the token test (Caso et al. 2012). However, this result should be taken with caution as, in this study; the analyses were performed by merging patients with FTD and AD.

In patients with VaD, one study did not find any correlation between cognitive deficits and brain oscillations, one study reported that reduced beta power is positively correlated with the MMSE score, as well as with the delay recall of the Rey Auditory Verbal Learning Test (RAVLT) (van Straaten et al. 2012). More studies would be needed to shed light on this correlation in VaD patients.

Possible mechanisms associated

The association between cognitive symptoms and changes in brain oscillations could be explained by cholinergic deficits that might account for both cognitive symptoms (Reisberg et al. 1982) and the slowing of EEG, especially in AD patients. In particular, alpha band is mainly modulated by global attentional readiness (Klimesch et al. 1998; Klimesch 1999). At rest, the alpha rhythms would be negatively correlated with cortical excitability and the

level of attention processes. For this reason, it has been suggested that the amplitude of the alpha rhythm and the corresponding cortical excitability reflect at least in part the time-varying inputs of the forebrain cholinergic pathways (Ricceri et al. 2004). However, it should be noted that studies investigating the relationship between cholinergic system and cognitive symptoms are old and are carried out primarily on animals. Therefore, further studies would be needed to clarify whether the dysfunction of the cholinergic system might account for the impairment in alpha activity and the cognitive deficits observed in dementia.

Changes in coherence and synchronization

In AD, reduced inter-hemispheric coherence has been found over the frontal, temporal, and parietal cortices in both alpha and beta frequency bands (Fonseca et al. 2011a,2011b; Knott et al. 2001; Musaeus et al. 2019; Zheng-Yan 2005) (Table 1c and d). Of note, another study reports an increase in parieto–occipital coherence in the alpha and beta frequency bands (Stam et al. 2006). These findings suggest a general long-range connectivity reduction in AD in favor of more local interactions, as reflected by an increase in connectivity in parieto–occipital regions. This might reflect loss of long-distance association's fibers (Stam et al. 2006). On the other hand, as parieto–occipital regions are relatively preserved in AD, the increase in connectivity in alpha and beta bands might reflect a compensation mechanism in a relatively healthy part of the network (Stam et al. 2006).

With respect to the lower frequency bands, a significant reduction of the intrahemispheric coherence in the delta band between central and occipital regions has been found (Adler et al. 2003; Knott et al. 2001; Zheng-Yan 2005).

Contrasting results have been reported for theta oscillations. Indeed, some studies found reduced intrahemispheric coherence between electrodes placed over parieto–occipital and temporo–occipital regions (Knott et al. 2000; Zheng-yan 2005). Other studies reported an increase in the intrahemispheric coherence between electrodes placed over the frontal areas (Musaeus et al. 2019; Sankari et al. 2011), over the parietal and central areas, and between temporo–central and temporo–parietal areas (Musaeus et al. 2019; Sankari et al. 2011). The discrepancy in the results might be due to differences in the sample sizes as well as to the medications status of the patients. Indeed, it has been shown that theta oscillations are modulated by the activity of the cholinergic system and by cholinergic therapy (Adler et al. 2003; Yener et al. 2007). On the other hand, abnormal frontal theta activity has been considered an index of a dysfunction of the hippocampo–fronto–parietal system during cognitive processes (Yener et al. 2007). We found only a previous study investigating coherence in FTD compared to AD (Yu et al. 2016). This study reported higher coherence in the delta and alpha frequency bands for FTD over frontal areas and increased posterior coherence in the theta band in AD. The authors concluded that these changes reflect the stronger impairment in the frontal areas of FTD compared to AD patients. However, further studies would be needed to identify the exact pattern of changes in coherence among these different types of dementia. Furthermore, we did not find recent studies investigating coherence in patients with VaD. Only a previous review reported a general disruption on long-distance coherence among several cortical areas (Jeong et al. 2001). However, the included studies were published before 2000. Recent studies are required to clarify to what extent vascular accidents might affect coherence.

With respect to synchronization (Tables 1e and f), abnormal synchronization in the alpha, theta, beta, and delta frequency bands have been reported in AD and VaD patients. In particular, delta and alpha synchronizations decrease in fronto–parietal regions in both AD and VaD patients (Babiloni et al. 2004b). In AD, the decrease in alpha synchronization also involves fronto–temporal regions (Babiloni et al. 2004b; Canuet et al. 2012; Stam et al. 2002, 2006).

Activity in the beta band decreases in fronto–temporal and fronto–parietal as well as in parietal and occipital areas in AD (Stam et al. 2002, 2006). In contrast, an increase in synchronization in the theta frequency band has been found in AD patients at central and parietal regions (Stam et al. 2006) as well as at right parieto–temporal regions and between left temporal and right prefrontal cortex (Canuet et al. 2012). However, these results are not in line with other studies reporting reduced theta synchronization (Babiloni et al. 2004b; Hata et al. 2016). As stated above, being theta oscillations strongly dependent on the cholinergic system, differences in the pharmacological status of patients included in the studies might account for the discrepancy in the results.

It is noteworthy that synchronization in the gamma band has been scarcely investigated in dementia. We found a previous study reporting a general reduction in gamma synchronization in AD (Stam et al. 2002). The author suggested that this reduction might be related to deficient or

Table 1c: Characteristics of studies exploring changes in EEG coherence in Alzheimer's diseases (AD), frontotemporal degeneration (FTD), and vascular dementia (VaD).

Study	Participants characteristics		Main results			
	Population	Comparator	Bands	Changes	Brain regions	Localization
Adler et al. (2003)	AD	Cognitively unimpaired	Delta	Decreased	Left centro–parietal, left temporal, right parieto–occipital	Intrahemispheric
			Theta	Decreased	Left temporal	Intrahemispheric
			Alpha	Decreased	Right centro–parietal, left temporal	Intrahemispheric
			Beta	Decreased	Left temporal	Intrahemispheric
Fonseca et al. (2011a)	AD	Healthy	Delta	No changes		
			Theta	No changes		
			Alpha	No changes		
			Beta	No changes		
Fonseca et al. (2011b)	AD	Healthy	Delta	No changes		
			Theta	No changes		
			Alpha	No changes		
			Beta	No changes		
Knott et al. (2000)	AD	Healthy	Delta	Decreased	Widespread (except for prefrontal–frontal)	Intrahemispheric
			Theta	Decreased	Right temporal	Intrahemispheric
			Alpha	No changes		
			Beta	No changes		
Musaeus et al. (2019)	AD	Healthy	Delta	Decreased	Left fronto–parietal, left parietal	Intrahemispheric
				Increased	Frontal	
			Theta	Increased	Widespread	Intrahemispheric
			Alpha	Decreased	Widespread	Intrahemispheric
			Beta	Decreased	Centro–occipital, centro–temporal	Intrahemispheric
				Increased	Frontal	
Sankari et al. (2011)	AD	Healthy	Delta	Increased	Left frontal, left temporo–frontal, left temporo–central, left temporo–parietal	Intrahemispheric
			Theta	Increased	Left frontal, left fronto–central, left temporo–central, left temporo–parietal	Intrahemispheric
			Alpha	Increased	Left frontal, left fronto–central, left temporo–central, left temporo–parietal, left parieto–central, left parieto–occipital	Intrahemispheric
			Beta	Increased	Left temporo–central, left parieto–central, left parieto–occipital	Intrahemispheric
Van Der Hiele et al. (2007)	AD	Healthy	Delta	No changes		
			Theta	No changes		
			Alpha	No changes		
			Beta	No changes		
Zheng-Yan (2005)	AD	Healthy	Delta	Decreased	Right centro–occipital	Intrahemispheric
			Theta	Decreased	Right parieto–occipital, left temporo–occipital	Intrahemispheric
			Alpha	Decreased	Right centro–parietal, right temporo–occipital	Intrahemispheric
			Beta	No changes		
Adler et al. (2003)	AD	Cognitively unimpaired depressive	Delta	Decreased	Central, occipital	Interhemispheric
			Theta	Decreased	Central, parietal, occipital	Interhemispheric
			Alpha	Decreased	Frontal, parietal, occipital	Interhemispheric
			Beta	No changes		
Fonseca et al. (2011a)	AD	Healthy	Delta	No changes		
			Theta	No changes		
			Alpha	Decreased	Frontal	Interhemispheric
			Beta	Decreased	Frontal, occipital	Interhemispheric
Fonseca et al. (2011b)	AD	Healthy	Delta	No changes		
			Theta	No changes		
			Alpha	Decreased	Frontal–temporal	Interhemispheric
			Beta	Decreased	Frontal–temporal	Interhemispheric

Table 1c: (continued)

Study	Participants characteristics		Main results			
	Population	Comparator	Bands	Changes	Brain regions	Localization
Knott et al. (2000)	AD	Healthy	Delta	Increased	Prefrontal	Interhemispheric
				Decreased	Central, parietal, temporal, occipital	Interhemispheric
			Theta	Decreased	Central, parietal, posterior–temporal	Interhemispheric
			Alpha	Decreased	Frontal, central, parietal, posterior–temporal	Interhemispheric
			Beta	No changes		
Musaeus et al. (2019)	AD	Healthy	Delta	Decreased	Fronto–parietal, fronto–temporal, pari-eto–occipital	Interhemispheric
			Theta	Increased	Widespread (with the exception of cz-Fz, Cz-T3, Cz-T5, Cz-T4)	Interhemispheric
			Alpha	Decreased	Widespread	Interhemispheric
			Beta	Decreased	Fronto–temporal	Interhemispheric
Van Der Hiele et al. (2007)	AD	Healthy	Delta	No changes		
			Theta	No changes		
			Alpha	No changes		
			Beta	No changes		
Yu et al. (2016)	AD	Subjective cognitive decline	Delta	Decreased (Vs. FTD)	Whole brain	
			Theta	Increased	Whole brain	
			Alpha	Decreased	Whole brain	
			Beta	No changes		
	FTD	Subjective cognitive decline	Delta	Increased	Whole brain	
			Theta	No changes		
			Alpha	No changes		
			Beta	No changes		
Zheng-Yan (2005)	AD	Healthy	Delta	No changes		
			Theta	No changes		
			Alpha	Decreased	Central, parietal, temporal	Interhemispheric
			Beta	No changes		

Table 1d: Characteristics of studies exploring changes in MEG coherence in Alzheimer's diseases (AD), frontotemporal degeneration (FTD), and vascular dementia (VaD).

Study	Participants characteristics		Main results			
	Population	Comparator	Bands	Changes	Brain regions	Localization
Stam et al. (2006)	AD	Healthy	Delta	No changes		Intrahemispheric
			Theta	No changes		Intrahemispheric
			Alpha	Decreased	Right parietal	Intrahemispheric
			Beta	Decreased	Left fronto–temporal	Intrahemispheric
Stam et al. (2006)	AD	Healthy	Delta	No changes		Interhemispheric
			Theta	No changes		Interhemispheric
			Alpha	No changes		Interhemispheric
			Beta	No changes		Interhemispheric

reduced information processing (Stam et al. 2002). However, recent evidence coming from animals' studies suggested that gamma oscillations might be altered in AD and that restoring this rhythm might induce an improvement of AD symptoms (Iaccarino et al. 2016). Therefore, future studies should also investigate the role of coherence in the gamma frequency band among different types of dementia also in light of new possible emerging treatments.

Table 1e: Characteristics of studies exploring changes in EEG synchronization in Alzheimer's disease (AD), frontotemporal degeneration (FTD), and vascular dementia (VaD).

Study	Participants characteristics		Main results			
	Population	Comparator	Bands	Changes	Brain regions	Localization
Babiloni et al. (2004b)	AD	Healthy	Delta	Decreased	Left fronto–parietal	Intrahemispheric
			Theta	Decreased	Right fronto–parietal	Intrahemispheric
			Alpha	Decreased	Midline fronto–parietal	Intrahemispheric
			Beta	No changes		
	VaD		Delta	Decreased	Right fronto–parietal	Intrahemispheric
			Theta	No changes		
			Alpha	Decreased	Midline fronto–parietal, right fronto–parietal	Intrahemispheric
			Beta	No changes		
Canuet et al. (2012)	AD	Healthy	Delta	No changes		
			Theta	Increased	Left fronto–temporal, right parieto–temporal	Intrahemispheric
			Alpha	Decreased	Medial fronto–temporal, medial parietal–temporal	Intrahemispheric
			Beta	No changes		
Hata et al. (2016)	AD	Healthy	Delta	Decreased	Widespread	Intrahemispheric
			Theta	Decreased	Right fronto–parietal	Intrahemispheric
			Alpha	No changes		
			Beta	No changes		
Babiloni et al. (2004b)	AD	Healthy	Delta	Decreased	Frontal, central, parietal	Interhemispheric
			Theta	Decreased	Frontal, central, parietal	Interhemispheric
			Alpha	Decreased	Frontal, central, parietal	Interhemispheric
			Beta	Decreased	Frontal, central, parietal	Interhemispheric
	VaD		Delta	Decreased	Frontal, central, parietal	Interhemispheric
			Theta	Decreased	Frontal, central, parietal	Interhemispheric
			Alpha	Decreased	Frontal, central, parietal	Interhemispheric
			Beta	Decreased	Frontal, central, parietal	Interhemispheric
Canuet et al. (2012)	AD	Healthy	Delta	No changes		
			Theta	Increased	Left temporal–central	Interhemispheric
			Alpha	No changes		
			Beta	No changes		
Hata et al. (2016)	AD	Healthy	Delta	Decreased	Widespread	Interhemispheric
			Theta	No changes		
			Alpha	No changes		
			Beta	No changes		

Possible mechanisms associated

Although the mechanisms underlying the reduction in long-range connectivity are still not clear, it has been suggested that they might be due to a decrease in acetylcholine (Fonseca et al. 2011a).

Overall, the loss of coherence in AD could be caused by the loss of anatomical connections between cortical areas (Stam et al. 2002). However, this hypothesis does not explain why changes regard mainly higher frequencies. A possible parallel explanation provided by previous studies is that the loss of cholinergic projections in specific regions, such as the nucleus basalis, would lead to stronger synchronization in lower frequencies and weaker synchronization in higher frequency bands (Villa et al. 2000).

Changes in synchronization in AD have been suggested to reflect anatomic and neuropharmacological abnormalities. Additionally, genetic factors such as the presence of the APOe4 gene might affect synchronization and should be further investigated.

Correlations between coherence, synchronization and cognitive processes

The studies revised here reported several associations between changes in coherence and synchronization and cognitive deficits. In particular, the reduction in the intrahemispheric alpha coherence is correlated with the immediate verbal recall score from the structured interview for the diagnosis of the Alzheimer's type of dementia

Table 1f: Characteristics of studies exploring changes in MEG synchronization in Alzheimer's disease (AD), frontotemporal degeneration (FTD), and vascular dementia (VaD).

Study	Participants characteristics		Main results			
	Population	Comparator	Bands	Changes	Brain regions	Localization
Stam et al. (2006)	AD	Healthy	Delta	No changes		Intrahemispheric
			Theta	Increased	Central, parietal	Intrahemispheric
			Alpha	Decreased	Fronto–temporal, left fronto–parietal	Intrahemispheric
			Beta	Decreased	Fronto–temporal, fronto–parietal	Intrahemispheric
Stam et al. (2002)	AD	Healthy	Delta	No changes		Intrahemispheric
			Theta	No changes		Intrahemispheric
			Alpha	Decreased	Temporal, right central, right frontal, right parietal	Intrahemispheric
			Beta	Decreased	Central, parietal, temporal, midline occipital	Intrahemispheric
Stam et al. (2006)	AD	Healthy	Delta	No changes		Interhemispheric
			Theta	No changes		Interhemispheric
			Alpha	No changes		Interhemispheric
			Beta	No changes		Interhemispheric
Stam et al. (2002)	AD	Healthy	Delta	No changes		Interhemispheric
			Theta	No changes		Interhemispheric
			Alpha	No changes		Interhemispheric
			Beta	No changes		Interhemispheric

(SIDAM) (Adler et al. 2003). Similarly, the fronto–temporal interhemispheric alpha coherence was correlated with the MMSE score and the visuospatial memory score in different studies from the Consortium to Establish a Registry for Alzheimer's disease (CERAD) (Fonseca et al. 2011b; Knott et al. 2000; Musaeus et al. 2019). In the frontal regions, reduced coherence in the alpha frequency band correlates with poorer performance at the MMSE (Knott et al. 2001), while reduced alpha interhemispheric occipital coherence correlates with the word list recall (Fonseca et al. 2011a). Fronto–temporal coherence in the beta band correlates with the word list recall and the visuospatial memory scores (Fonseca et al. 2011b).

The involvement of alpha rhythms in memory has previously been reported in studies conducted in healthy subjects (Klimesch 1999). It has been proposed that alpha rhythm would be modulated mainly by thalamo–cortical and cortico–cortical interactions that influence the information transmission between cortical and subcortical streams and the retrieval of semantic information from the brain (Pfurtscheller and Lopes Da Silva 1999).

A negative correlation has been found between MMSE and the delta coherence of the left brain hemisphere (Fonseca et al. 2011b). The interhemispheric coherence also showed positive correlation with word list recall and praxis recall (Fonseca et al. 2011b).

However, other studies did not find correlations between the synchronization in any frequency band and the MMSE scores in AD (Adler et al. 2003; Stam et al. 2002; van der Hiele et al. 2007).

A negative correlation was found between delta frontal–temporal–central–parietal coherence with the Boston naming test in the AD group (Fonseca et al. 2011b). On the other hand, the occipital interhemispheric coherence in the theta band showed positive correlation with word list recall (Fonseca et al. 2011b).

Regarding synchronization, reduced alpha synchronization in the frontal and temporal regions correlates with the MMSE scores (Stam et al. 2006). Similarly, performance in the MMSE is positively correlated with beta synchronization in temporal regions (Stam et al. 2006), whereas the correlation becomes negative with the reduction of theta synchronization in the same areas (Canuet et al. 2012). Interestingly, a previous study reported that higher connectivity in the delta frequency band between the anterior cingulate cortex, the frontal eye fields, the hippocampus, and the parietal lobule is positively correlated with the MMSE score (Hata et al. 2016). In particular, the higher the connectivity the better the cognitive performance. These regions are involved in several cognitive processes (Collette and Van Der Linden 2002). Thus, the abnormal synchronization between these brain regions might reflect the disintegration of connectivity between these brain areas and might account for cognitive deficits of AD (Hata et al. 2016).

Possible mechanisms associated

From a physiopathological point of view, the association of reduced coherence in alpha band with worse performance

could be the result of reduced functional connection between areas supporting the hypothesis of AD as a disconnection syndrome (Delatour et al. 2004). Thus, the loss of the cortico–cortical association fibers needed for functional connectivity or the reduced cholinergic coupling interaction between cortical neurons might be two mechanisms responsible for the reduction of coherence in the alpha band and for cognitive symptoms. In case of slower frequency bands, the association between delta and theta coherence and cognitive deficits might be due to a decrease in acetylcholine in AD (Locatelli et al. 1998).

Reduced cortical synchronization in patients with AD might be caused by the disruption of axonal connections between many brain areas due to the presence of neurofibrillary tangles and amyloid plaques, disrupting the normal electrical communication of the brain thus inducing cognitive difficulties (Sankari et al. 2011). However, it is not clear why the reduced connectivity involves some brain rhythms whereas others are not affected. Investigating the connectivity loss could be useful in the differential diagnosis of dementia and provide insights into the pathophysiological mechanisms underlying cognitive deficits in each type of dementia. This holds especially for VaD and FTD whose connectivity patterns have not been investigated yet.

Amplitude and latency changes in ERPs

ERP components have been investigated in dementia mostly by using the auditory oddball task. Visual paradigms were instead used to assess ERP only in AD.

AD patients exhibit a reduced peak amplitude and longer latencies of the P300 over the frontal, central, and parietal areas during the presentation of tones (Ally et al. 2006; Ashford et al. 2011; Caravaglios et al. 2010, 2008; Chang et al. 2014; Frodl et al. 2002; Gironell et al. 2005; Hirata et al. 2000; Juckel et al. 2008; Lai et al. 2010; Lee et al. 2013; Muscoso et al. 2006; Sumi et al. 2000). Longer latencies have also been reported for the N100 and N200 (Bennys et al. 2007; Caravaglios et al. 2008; Chang et al. 2014). However, other studies have not reported changes in the amplitude of P300, P200, and N100 (Caravaglios et al. 2008) nor differences in the latencies of N200, N100, and P200 (Caravaglios et al. 2008; Chang et al. 2014; Lai et al. 2010).

Additionally, we found only two studies investigating ERP in VaD reporting that, in response to auditory stimuli, P300 has longer latencies than in healthy subjects and shorter latencies than in AD (Muscoso et al. 2006; Yamaguchi et al. 2000).

ERPs in response to visual stimuli were scarcely investigated. Overall, a significantly longer P300 latency for visual stimuli has been reported in AD patients (Kurita et al. 2010), whereas we did not find studies investigating such components in VaD and FTD patients (Table 1g).

Possible mechanisms associated

The cholinergic deficit and the decrease in dopamine transmission could induce modification of ERP components (Hansenne et al. 2000). For instance, P300 is mainly generated over temporo–parietal areas (Chang et al. 2014; Juckel et al. 2008; Smith et al. 1990) whose activity is modulated by cholinergic therapies (Frodl et al. 2002) and the reduction of its amplitude is in line with the degeneration of neurons in temporal lobes exhibited by patients with AD.

Further consideration is needed taking into account the timing of each ERP. It has been suggested that the absence of changes in the N100 and the P200 is probably due to the fact that these components reflect a sensorial analysis of the stimulus that is relatively preserved in AD. In contrast, later components such as the P300 and the N200 mostly reflect a cognitive level of stimulus processing that is instead impaired in AD (Caravaglios et al. 2008). The prolonged latencies in these ERP probably imply that more time is needed for information processing due to a pathological slowing of the process of stimulus evaluation (Kutas et al. 1977). We did not find any study analysing ERP changes in FTD. Further studies should address this topic to better clarify the potential role of ERP as a marker for a differential diagnosis between types of dementia.

Correlations between ERPs and cognitive processes

Each ERP component has specific amplitude associated with the intensity of a specific cognitive process and a latency related to the processing time needed for the task (Sur and Sinha 2009). Early ERPs are related to attention selection mechanisms whereas later ERP are related to stimuli processing (Katada et al. 2005). Changes in ERP amplitude and latency during cognitive tasks have been found in both normal aging (Ghani et al. 2020) and dementia (Polich and Corey-Bloom 2005). In particular, some previous studies investigated the correlation between specific ERP components and neuropsychological tests and overall reported a negative correlation between P300 latency and MMSE scores (Bennys et al. 2007;

Table 1g: Characteristics of studies exploring changes in ERP in Alzheimer's disease (AD), frontotemporal degeneration (FTD), and vascular dementia (VaD).

Study	Participants characteristics		Main results		
	Population	Comparator	ERP	Latency	Amplitude
Ally et al. (2006)	AD	Healthy	P300	Longer (trend)	Decreased
Ashford et al. (2011)	AD	Healthy	P300	No changes	Decreased
Bennys et al. (2007)	AD	Healthy	N200	Longer	No changes
			P300	Longer	Decreased
Caravaglios et al. (2008)	AD	Healthy	N100	No changes	No changes
			N200	Longer	No changes
			P200	No changes	No changes
			P300	Longer	No changes
Caravaglios et al. (2010)	AD	Healthy	N200	Longer	No changes
			P300	Longer (trend)	Decreased (trend)
Chang et al. (2014)	AD	Healthy	N100	No changes	No changes
			N200	Longer	No changes
			P200	No changes	No changes
			P300	Longer	No changes
Frodl et al. (2002)	AD	Healthy	P300	Longer	Decreased
Gironell et al. (2005)	AD	Healthy	P300	Longer	No changes
Hirata et al. (2000)	AD	Healthy	N100	Longer	Decreased
			N200	Shorter	No changes
			P200	No changes	No changes
			P300	Longer	Decreased
Juckel et al. (2008)	AD	Healthy	P300	Longer	Decreased
Lai et al. (2010)	AD	Healthy	N100	No changes	No changes
			N200	No changes	No changes
			P200	No changes	No changes
			P300	Longer	No changes
Lee et al. (2013)	AD	Healthy	P300	No changes	Decreased
Muscoso et al. (2006)	AD	Healthy	N100	Longer	
			N200	Longer	
			P200	Longer	
			P300	Longer	
	VaD	Healthy	N100	No changes	
			N200	No changes	
			P200	No changes	
			P300	Longer	
Sumi et al. (2000)	AD	Healthy	N100	No changes	
			N200	Longer	
			P200	No changes	
			P300	Longer	
Yamaguchi et al. (2000)	AD	Healthy	P300	Longer	No changes
	VaD	Healthy	P300	Longer	Decreased
Kurita et al. (2010)[a]	AD	Healthy	N100	No changes	
			N200	No changes	
			P200	No changes	
			P300	Longer	

[a]this study investigated visual event-related potentials.

Hirata et al. 2000; Lee et al. 2013). Similarly, in AD and VaD, P300 is negatively correlated with intelligence scales (Yamaguchi et al. 2000).

Furthermore, in patients with AD, P300 latency is positively correlated with the part "A" of the trail making test (TMT-a) (Lee et al. 2013) as well as with the world list recognition test, word fluency for executive functions test, and with scores in visuospatial memory, visuospatial abilities measured using CERAD-K (Lee et al. 2013). N200 and P300 latencies have been found to positively correlate

with performance obtained in the Stroop test and in the TMT (Bennys et al. 2007).

Although the neuropsychological origin and meaning of P300 has not yet been fully clarified, P300 appears when a stimulus requires attention to the frontal lobe and its activity ends when attention resources are allocated for stimulus evaluation and subsequent memory updating (Polich 2007). In general, current evidence suggests that, in AD patients, P300 is significantly correlated with executive function, memory, and visuospatial functions (Lee et al. 2013). In other words, early changes of ERP, especially P300 and N200 could reflect the deficit of attentional processes linked to working memory and dysfunctional frontal processing in AD (Baddeley et al. 2001; Sebastian et al. 2006). In this view, the non-invasive examination of ERP might contribute to the early assessment of cognitive deficits. Thus, more studies would be needed investigating ERP changes in FTD and VaD patients to support with good sensitivity and specificity the diagnosis of dementia.

Conclusions and future perspectives

The present work revised evidence of EEG and MEG oscillatory changes as well as ERP changes in dementia. Overall, the current literature suggests that changes in frequency power, synchronization and in ERP constitute possible markers of dementia. In particular, changes in brain oscillations and ERP might be to some extent informative regarding the cognitive status of the patients and may add informative value to other pathophysiological markers, such as amyloid tracer PET scans, T-tau, P-tau, and neuroimaging techniques.

However, several important issues emerged from the review of the current literature, which constitute potential avenues for future research. First, very few studies investigated gamma oscillations in dementia. Recent studies have highlighted the role of modulating gamma oscillations in both healthy subjects and patients (Adaikkan and Tsai 2020; Giustiniani et al. 2019, 2021). In particular, restoring gamma oscillations reduces beta amyloid and tau protein in mice with induced AD (Iaccarino et al. 2016). Due to the growing interest in the therapeutic effect of gamma oscillations and the role of their alterations in cognitive symptoms, more studies would be needed to investigate the role of these oscillations in dementia. Second, only a few studies have been conducted in VaD and FTD patients. This lack of studies limits the generalizability of the conclusion in these groups of patients. Finally, the role of neurotransmitters, such as acetylcholine and GABA, must be better clarified by conducting new studies in healthy participants and patients with dementia. Thus, more studies would be needed to investigate and compare brain oscillations and underlying mechanisms especially in patients with FTD and VaD.

Overcoming these issues, would allow a more precise mapping of the topography of electrophysiological changes and of the underlying mechanisms. This in turn, could support the treatment, for instance, through the application of non-invasive brain stimulation techniques (Turriziani et al. 2012). In each pathology, specific regions could be a target for therapeutic approaches that aim to improve cognitive symptoms and restore brain oscillations.

In conclusion, current evidence from EEG and MEG studies supports their use as valid techniques to investigate alterations in brain rhythms but suggests the need to further investigate this topic. Changes in frequency power, coherence, and ERPs could be considered objective markers to employ in clinic environments and to combine with traditional biomarkers and with neuropsychological assessment. Furthermore, these EEG/MEG measurements could also aid clinical decision making and could be used as starting points for the development of new rehabilitative treatments targeting brain rhythms or ERP components.

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